



Tetralogy of Fallot in a 2-year-old Holstein heifer

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Abstract — A 2-year-old, purebred Holstein heifer with exercise intolerance and cardiovascular compromise was diagnosed at postmortem with tetralogy of Fallot, which typically results in death within a few months of life. Survival past the age of 2 was unexpected. The concurrent endocarditis of the pulmonic valve is discussed.

Résumé — **Tétralogie de Fallot chez une génisse Holstein âgée de 2 ans.** Un diagnostic post mortem de tétralogie de Fallot a été posé sur une génisse Holstein de race pure âgée de 2 ans présentant une intolérance à l'exercice et un dysfonctionnement cardiovasculaire. Dans cette condition, la mort survient habituellement dans les premiers mois de la vie et une survie de plus de 2 ans est inattendue. L'endocardite concomitante de la valvule pulmonaire est décrite.

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A 24-month-old Holstein heifer was presented to the Ambulatory Service of the Atlantic Veterinary College (AVC) with a past history of poor growth and inability to conceive, and with a recent history of exercise intolerance. The animal was a singleton and, therefore, unlikely to be a freemartin; up until recently, it had not exhibited any other significant health problem.

On visual examination, the animal demonstrated exercise intolerance by becoming dyspneic following mild exertion. Bilateral distension of the external jugular vein and a grade 6/6 holosystolic murmur were found on physical examination. The murmur was auscultable bilaterally, but the point of maximal intensity was over the base of the heart on the left side. Auscultation of lung fields revealed crackles consistent with pulmonary edema or pneumonia. At this point, the heifer was referred to the Veterinary Teaching Hospital of the AVC for further evaluation.

On physical examination, the heifer was deemed to be small for her age, but in good body condition (456 kg body weight (BW)). She was febrile (rectal temperature 39.8°C), tachycardic (96 beats/min), and tachypneic (90 breaths/min). A complete blood cell count revealed evidence of polycythemia with a mild elevation in the hematocrit (0.472 L/L; normal, 0.24 to 0.46 L/L) and hemoglobin concentration (167 g/L; normal, 80 to 150 g/L), while the total protein was normal (77 g/L; normal 60 to 85 g/L). The leukogram and fibrinogen were within normal limits. The heifer was treated once with 75 mg of furosemide (Lasix; Hoechst Canada, Montreal,

Quebec) for right-sided heart failure and pulmonary edema, but no improvement was noted. Echocardiography was performed using an HDI 3000 ultrasound unit with a 3–2 MHz probe (ATL Diagnostics, Bothell, Washington, USA). The examination was incomplete, as the great vessels and semilunar valves were not visualized; but an enlarged right atrium and a small to normal left ventricle were seen. Based upon these limited findings of right-sided cardiac enlargement with a left-sided murmur, the initial differential diagnosis included atrial septal defect, pulmonic valvular stenosis, or valvular endocarditis of the tricuspid valve leading to valvular insufficiency.

Because of the poor prognosis, no further diagnostics were performed. The heifer was euthanized and necropsied. The heart was moderately enlarged and globose. The right ventricular free wall was moderately thickened with a left to right wall ratio of 1.5:1, in contrast to the normal 3:1. There was infundibular pulmonic stenosis, and the pulmonic valve had a proliferative, fibrosuppurative mass that almost completely occluded the lumen of the pulmonary artery. A high (4 cm) ventricular septal defect was present and the aorta overrode the left and right ventricles in the area of the septal defect, which was consistent with tetralogy of Fallot (TOF). The lungs were also moderately congested.

Tetralogy of Fallot results from abnormal development of the conal septum in the embryonic heart, which narrows the right ventricular infundibulum (7). This narrowing of the infundibulum leads to pulmonary stenosis. Another result of the infundibular defect is an inability of the conal septum to participate in closure of the interventricular foramen, leaving a septal defect and overriding aorta. The 4 components of TOF are a ventricular septal defect (VSD), overriding of the interventricular septum by the aorta, pulmonic stenosis, and a compensatory hypertrophy of the right ventricle (2).

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This combination of defects results in blood being shunted from the right ventricle through the ventricular septal defect into the aorta, when right ventricular outflow obstruction is significant (1). The right ventricular outflow obstruction is due to the pulmonic stenosis, which increases pressure in the right heart significantly to overcome the normally higher pressure in the left ventricle. This right to left shunt is facilitated by the presence of the VSD, which if present without the other defects would typically cause a left to right shunt. In TOF, clinical signs include exercise intolerance and cyanosis from hypoxia due to the right to left shunting of blood and right-sided heart failure.

In cases of TOF, auscultation usually reveals high-pitched systolic ejection murmur of pulmonic stenosis or a harsh holosystolic murmur of VSD (2). Occasionally, an accentuated 4th heart sound (S4) may be heard. A 4th heart sound is commonly associated with atrial contraction, which is more apparent in TOF due to the atria contracting against increased resistance in the right ventricle caused from increased filling pressure (2). The murmur of an isolated VSD is usually bilateral with a point of maximal intensity over the apex of the heart on the right side. However, in TOF, due to the increased pressure in the right ventricle, the point of maximal intensity is on the left side. Principle features of the diagnosis of TOF with echocardiography are a large aorta that overrides the interventricular septum and a discontinuity between the interventricular septum and the aorta (3). Other features that may be present include thickening of the right ventricular free wall or septum, dilatation of the right ventricular chamber, and narrowing of the right ventricular outflow tract (3). Doppler studies have documented that right ventricular outflow obstructions are also important features of the malformation. In this case, no Doppler evaluation was performed. However, on cardiac ultrasonography, the free wall of the right ventricle was thickened, although not as dramatically as in other cases in which the right and left ventricular walls were found to be equal in thickness (2). Technical difficulties associated with echocardiography in dyspneic cows may be the reason that it was difficult to ascertain pathology on echocardiography.

Of particular interest in this case was the fact that the heifer had lived to 2 y of age with TOF. Although she was smaller than her pen-mates, the only concern up until the period of exercise intolerance was that she had not yet conceived. Based on a review of the pertinent literature (2–6), this is the first time that a case has been reported where a cow has lived to this age. Perhaps in this case, the degree of cardiovascular compromise due to the mild stenosis of the pulmonic artery was less severe than in the other cases that have been reported (2–6), or perhaps the VSD was not large enough to allow for nonrestricted right to left shunting. This is similar to a

case reported by Hare et al (4), but that animal only survived to 10 mo of age before evidence of cardiovascular compromise was seen. The heifer in this current report was likely compensating for her TOF until the right ventricular outflow was further compromised by the pulmonic valvular endocarditis. Evidence of the heifer's ability to compensate temporarily was illustrated by the fact that in her case, only a mild polycythemia was present, whereas in a chronic case with a cyanotic animal, a higher degree of polycythemia would be expected.

The occurrence of pulmonic valvular endocarditis in association with the pulmonic stenosis is also worth noting, since most cases of endocarditis involve adult cows and affect the tricuspid valve (7). Unfortunately, the etiology of the endocarditis was not established, since bacteriological blood cultures were not done and presumptive attempts to determine bacterial etiology on histological sections were not made. Possibly, endothelial damage caused by increased turbulence and the stenotic pulmonic lesion predisposed this heifer to endocarditis of the pulmonic valve, similar to what has been observed in young swine with subaortic stenosis (8). Due to the age of the animal, TOF was not included in the initial differential diagnosis. Although rare, TOF should remain on the differential list when a young adult bovine presents with evidence of cardiac failure and has a bilateral systolic murmur with a point of maximal intensity over the base of the heart on the left side.

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